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Original Article

Echocardiographic parameters in clinical responders to surgical pericardiectomy – A single center experience with chronic constrictive pericarditis

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ABSTRACT

Background: Chronic constrictive pericarditis (CCP) is the end result of chronic inflammation of the pericardium. Developing countries continue to face a significant burden of CCP secondary to tuberculous pericarditis. Surgical pericardiectomy offers potential cure. However, there is paucity of echocardiography data in post-pericardiectomy patients vis-a-vis their clinical status. We studied the changes in multiple echocardiographic parameters in these patients before and after pericardiectomy.

Methods: Twenty-three patients (14 men, 9 women) who underwent pericardiectomy for CCP in the last 5 years (from January 2009 to December 2014) were subjected to detailed clinical and echocardiographic evaluation during the study period (between June 2013 and December 2014). Patients with residual symptoms of NYHA class II and below were considered as 'responders'. The data thus obtained were compared to the pre-operative parameters.

Results: After pericardiectomy, the incidence of vena caval congestion decreased from 100% to 15% ($p < 0.001$). There was significant reduction in the mean left atrial size from 39.33 $\pm$ 10.52 mm to 34.45 $\pm$ 10.08 mm ($p < 0.001$) and also the ratio of left atrium to aortic annulus from 1.93 to 1.69 ($p < 0.001$) among 'responders' to pericardiectomy. Septal bounce was observed to persist in 5 (25%) patients after pericardiectomy. There was significant respiratory variation of 39.23 $\pm$ 15.11% in the mitral E velocity before pericardiectomy. After pericardiectomy, this variation reduced to 14.43 $\pm$ 7.76% ($p < 0.001$). There was also significant reduction in the respiratory variation in tricuspid E velocities from 31.33 $\pm$ 18.81% to 17.35 $\pm$ 16.26% ($p < 0.001$). After pericardiectomy, the mean ratio of mitral annular velocities, medial e′: lateral e′, reduced from 1.08 to 0.87 ($p < 0.03$). The phenomenon of 'annulus reversus' was found to persist in 6 'responders', thereby reflecting a 50% reduction in its

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incidence after pericardiectomy ($p < 0.001$). The ratio of mitral E to medial e′ (E/e′) increased from 4.21 ± 1.35 before pericardiectomy to 6.91 ± 2.62 after pericardiectomy ($p = 0.001$).
Conclusion: Among clinical responders to surgical pericardiectomy, echocardiographic assessment revealed a significant reduction in vena caval congestion, LA size, ratio of LA to aortic annulus, septal bounce, respiratory variation in mitral and tricuspid E velocities, mitral annular medial e′ and the phenomenon of annulus reversus. Also, there was a significant rise in minimum tricuspid and mitral E velocities and the E/e′ ratio.

1. Background

Chronic constrictive pericarditis (CCP) is an uncommon but disabling disease. It results from chronic pericardial inflammation and fibrosis. As many as 30–60% of patients with tuberculous pericarditis develop CCP as sequelae.[1] It is of substantial clinical interest because of the potential for surgical cure.[2] Pericardiectomy is the only accepted curative measure, which provides dramatic clinical improvement. However, there is paucity of Indian data on echocardiographic parameters vis-a-vis the clinical outcome in patients post-pericardiectomy. We assessed the echocardiographic parameters in these patients and compared it with their pre-procedural status.

2. Methods

2.1. Study population

Over an 18-month period from June 2013 to December 2014, we could access 23 patients who underwent pericardiectomy at the department of Cardiothoracic Surgery of our institute within the last 5 years and had adequate pre-procedural records. These patients were included in our study and underwent clinical and echocardiographic assessment during the study period. The pre- and post-pericardiectomy data thus obtained were compared.

2.2. Diagnosis of CCP

Presence of typical clinical, CT and echocardiographic findings was considered to be diagnostic of CCP (Table 1). Diagnosis was ultimately confirmed at the time of surgery in all patients. Cardiac catheterization was performed in three patients due to initial diagnostic ambiguity.

2.3. Clinical parameters

The clinical parameters considered in this study were the symptoms of dyspnea and fatigability as per the New York Heart Association (NYHA) classification, chest pain, edema, and ascites. The presence of clinical signs such as distended neck veins, Kussmaul sign, pulsus paradoxus, peripheral edema, and pericardial knock were also included in assessment. Patients having symptom class of NYHA II or below at post-operative evaluation were considered as 'responders' to pericardiectomy.

2.4. Echocardiographic examination

Echocardiography was done at least 15 days pre-operatively in all patients and post-operatively over 3 months to 6 years (a median of 9 months). All patients had comprehensive evaluation on the Philips iE33 (Philips Medical Systems, Andover, MA, USA) commercially available echocardiography system using a 1.6 MHz transducer (S5-1, Philips Medical Systems, Andover, MA, USA). M-mode, 2D, pulsed wave (PW) Doppler, and tissue Doppler Imaging (TDI) interrogation were performed both before and after pericardiectomy. Left ventricle (LV) ejection fraction (EF) was calculated by 2D echocardiography with a modification of the method by Quinones et al.[3] Right ventricular (RV) function was assessed by eye-balling and TAPSE (tricuspid annular peak systolic excursion). Mitral

Table 1 – Clinical and imaging features of chronic constrictive pericarditis.

Clinical
Ascites Precox
Peripheral odema
Easy fatigability
Dyspnea
Atypical chest pain
Engorged neck veins
Kussmaul sign
Prominent *x* and *y* descents of jugular venous pulse
Pulsus paradoxus (uncommon)
Apical retractions
Pericardial knock
Chest radiograph
Pericardial Calcification
Pericardial effusion
Echocardiography
Congested inferior vena cava without any respiro-phasic variation
Thickened or calcified pericardium
Septal bounce
Pericardial effusion
More than 25% expiratory increase in mitral inflow (E) velocity
Annulus reversus
Mediastinal CT
Pericardial calcification and thickness > 3–4 mm

and tricuspid valvular regurgitation was assessed semi-quantitatively as grade 1+ to 4+ with color flow imaging.

The size and respirophasic variation of the inferior vena cava (IVC) were assessed using the M-mode in subcostal view. Enhanced ventricular interdependence, a key hemodynamic feature of CCP, leads to 'septal bounce' and exaggerated respiratory variation in mitral and tricuspid inflow velocities, which are 180° out of phase. Septal bounce was detected on M-mode as paradoxical bouncing motion of the interventricular septum initially directed toward and then away from the LV during early diastole. Mitral and tricuspid inflow velocities were obtained by PW Doppler interrogation in apical 4-chamber (A4C) view with sample volume of 2–4 mm. Gains were minimized to allow for a clear tissue signal with minimal background noise. All Doppler velocities were recorded with a chart recorder at a sweep speed of 25 or 75 mm/sec. The velocities measured include mitral and tricuspid peak velocity of early (E) and late (A) filling. The peak E velocities were obtained at the mitral and tricuspid inflow regions in both the phases of respiration (assessed clinically) (Fig. 1). Peak annular velocities were measured using TDI in A4C in early diastole (e′) with sample volume of 2–4 mm placed at the septal (medial e′) and lateral (lateral e′) corner along the mitral annular plane (Fig. 2). In patients with atrial fibrillation, 10 consecutive signals were measured and averaged.

2.5. *Operative details*

Pericardiectomy was performed through midline sternotomy in all patients. Cardiopulmonary bypass (CPB) was not routinely used and was required in only two patients due to intraoperative hypotension. The standard protocol for pericardial resection at our institution is total pericardiectomy with removal of the anterior pericardium (between the phrenic nerves), posterior pericardium (left of the phrenic nerve) and also the diaphragmatic component. At sites, where the constricting peel was highly adherent and calcific, islands of pericardium were left behind after taking multiple incisions. This was to avoid injury to the atria, major vessels and coronaries. Intraoperative central venous pressure (CVP) monitoring was performed. No patient underwent concomitant coronary arterial bypass grafting or valvular surgery. Visceral pericardiectomy was performed as and when required.

2.6. *Statistical analysis*

Descriptive data are reported as mean ± SD or count (percent), as appropriate. The Shapiro–Wilk's test was used to check the normality of the outcome distributions. Paired *t* tests or Wilcoxon signed rank tests (for non-normal data) were used to

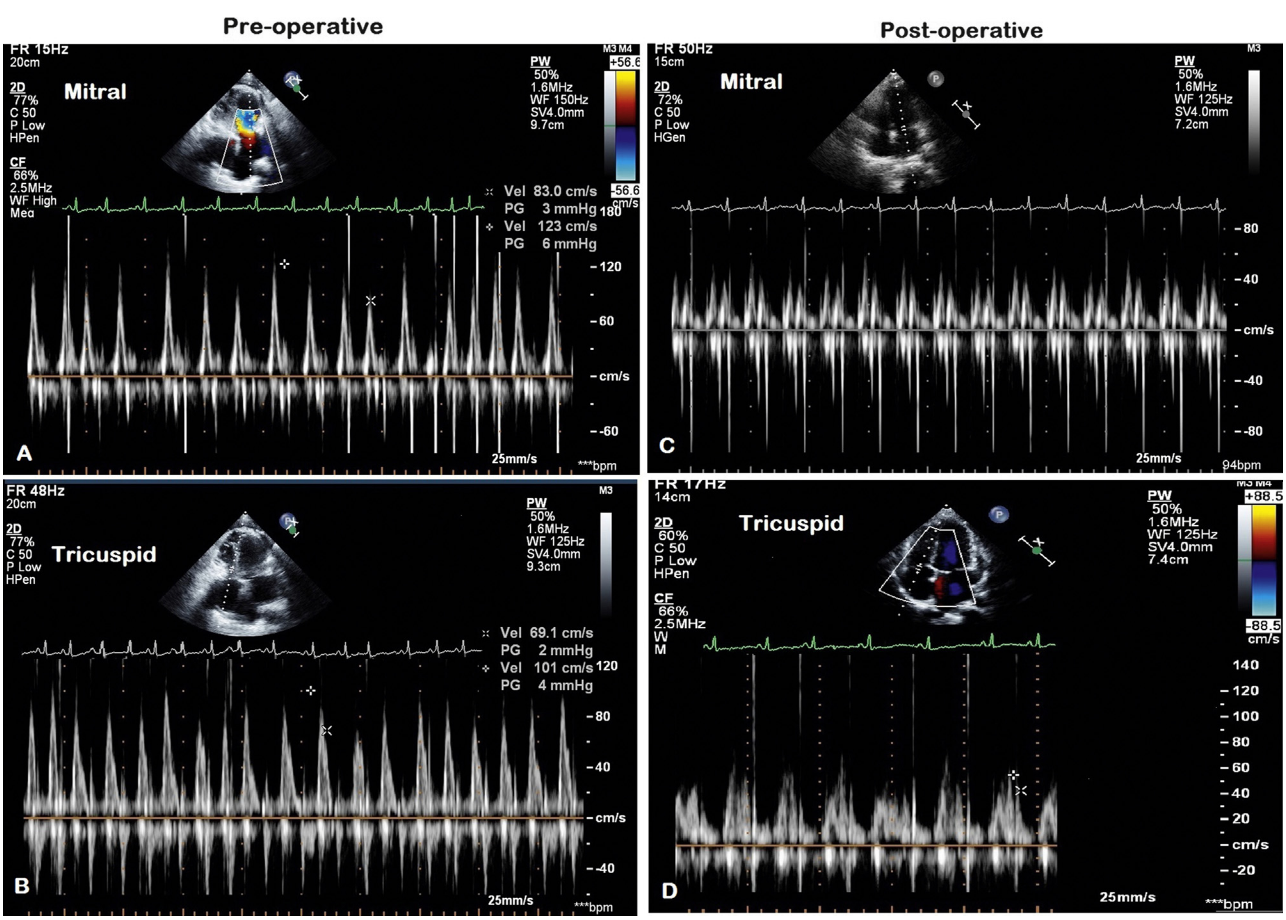

Fig. 1 – Image of the pulsed wave (PW) Doppler signals at the (A, C) mitral inflow and (B, D) tricuspid inflow region in a patient showing significant respiratory variation (A, B) pre-operatively as compared to the (C, D) post-operative PW Doppler signals.

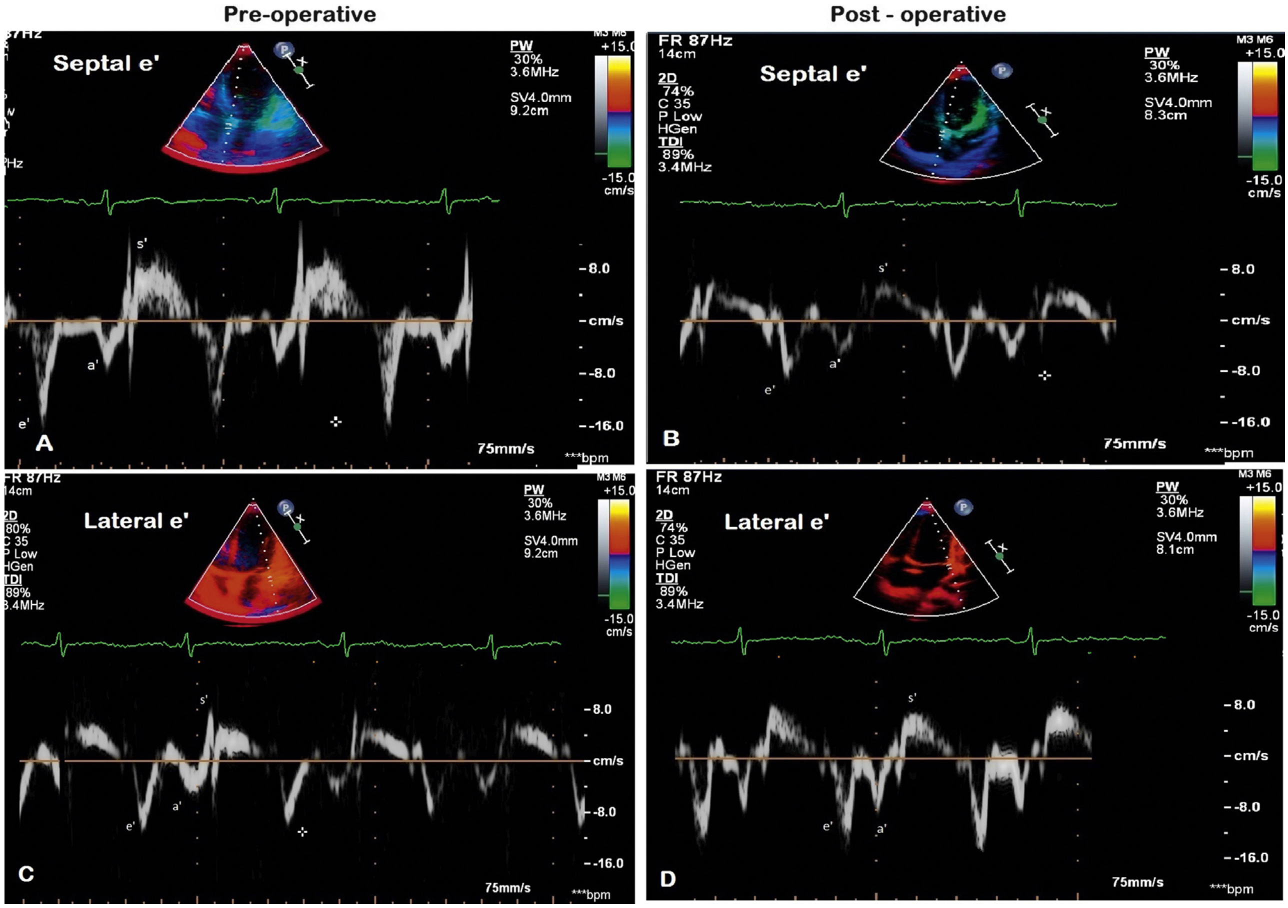

Fig. 2 – Image of Doppler signals using Tissue Doppler Imaging (TDI) in apical 4-chamber view with 2–4 mm sample volume placed at the septal corner along the mitral annular plane of a patient showing (A) pre-operative and (B) post-operative change in early diastole (e′) velocities (septal e′). Similarly TDI at lateral corner along the mitral annular plane in the same patient shows improvement in early diastole (e′) velocities (lateral e′) (D) post-operatively as compared to the (C) pre-operative image. As the septal e′ is larger than lateral e′, the phenomenon of 'annulus reversus' is also demonstrated in the pre-operative images.

assess the echocardiographic parameters before and after pericardiectomy. Differences were considered statistically significant at $p \leq 0.05$.

3. Results

3.1. *Patient characteristics*

Amongst the 23 patients studied, 20 patients (12 men, 8 women) had residual symptom class of NYHA II and below and were therefore considered as 'responders'. Three patients (2 men, 1 woman) continued to remain in NYHA class III after pericardiectomy and were considered as 'non-responders'. Further analysis was done only in the 'responders' group. The age of patients in this group ranged from 12 to 56 years (mean of 32.9 ± 15.43 years). All patients received 6 months of multi-drug anti-tubercular treatment as per institutional policy. However, a histopathological diagnosis of tubercular pericarditis was made in 13 (65%) patients and the remaining 7 patients were labeled as non-specific chronic pericarditis (Fig. 3). One patient in the latter group had developed symptomatic CCP, 12 years after surgical closure of a secundum atrial septal defect. All responders were in sinus rhythm.

3.2. *Clinical features*

Almost all the patients were in NYHA class III or IV prior to pericardiectomy. Fatigability and abdominal distension were the primary complaints in all patients. Venous distension and positive Kussmaul sign were seen in all patients. Clinical characteristics prior to surgery are shown in Table 2.

3.3. *Radiologic findings*

The chest radiograph demonstrated pericardial calcification (Fig. 4) in 20% of patients, while mean pericardial thickness measured using mediastinal CT was 5.3 ± 1.1 mm.

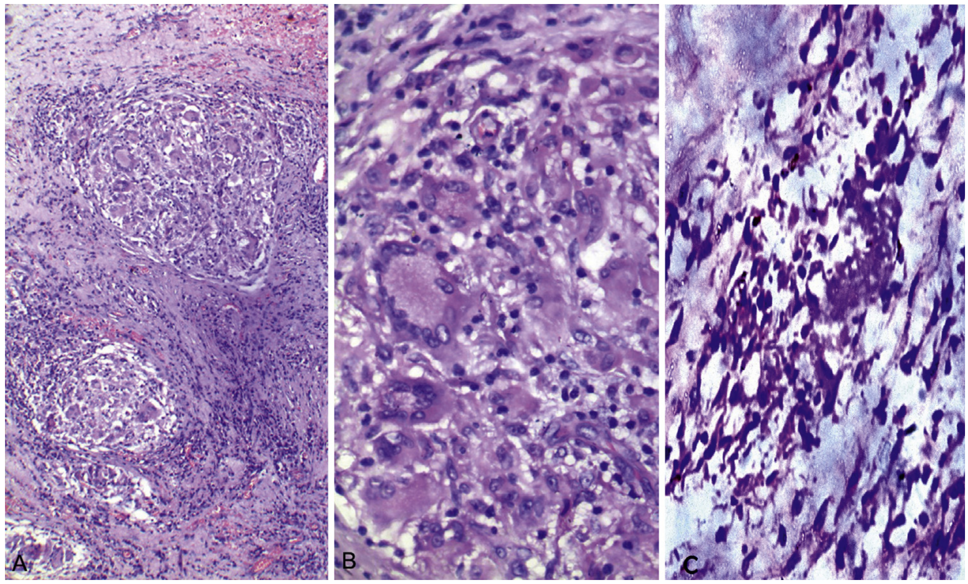

Fig. 3 – Photomicrograph of H&E specimen of (A: 250×, B: 400×) tuberculous pericarditis demonstrating thickened pericardium with multiple loosely formed epithelioid cell granulomas and Langhan's giant cells as compared to (C: 400×) chronic non-granulomatous inflammatory pericarditis.

3.4. *Echocardiography and Doppler findings*

The comparative echocardiographic findings, pre- and post-pericardiectomy in the 'responders' are summarized in Table 3.

Table 2 – Pre-procedural clinical data of patients with chronic constrictive pericarditis who responded to pericardiectomy.

	Pre-pericardiectomy (%)	
		%
Symptoms		
Fatigability		
NYHA IV	2	10
NYHA III	17	85
NYHA III	1	5
NYHA I	0	0
NYHA (mean)	3.1 ± 0.3	
Dyspnea	13	65
Atypical chest pain	5	25
Abdominal distension	20	100
Symptom duration (months)	12.3 ± 5.1	
Signs		
Distended neck veins	20	100
Kussmaul sign	20	100
Pulsus paradoxus	9	45
Peripheral odema	9	45
Pericardial knock	12	60
Hepatomegaly	17	85
Ascites	18	90
Chest radiography		
Pericardial calcification	7	35
CT pericardial thickness (mm)	5.3 ± 1.1	
Anti tubercular treatment	20	100

After pericardiectomy, there was significant reduction in the incidence of IVC congestion ($p < 0.001$) as well as the left atrial (LA) size from 39.33 ± 10.52 mm to 34.45 ± 10.18 mm ($p < 0.001$). Septal bounce was found to persist in 5 (25%) patients. No patient had residual pericardial effusion or ventricular dysfunction.

There was a significant rise in the inspiratory mitral E velocity from 51.90 ± 15.63 cm/sec to 70.30 ± 18.35 cm/sec ($p < 0.001$). Conversely, there was a significant rise in the expiratory tricuspid E velocity from 37.10 ± 7.90 cm/sec to 47.75 ± 11.15 cm/sec ($p < 0.001$). However, there was no significant change in the expiratory mitral E and inspiratory tricuspid E velocities (p values were 0.46 and 0.806, respectively). Thus, there was a rise in the minimal E velocities (i.e. during inspiration at mitral valve and expiration at tricuspid

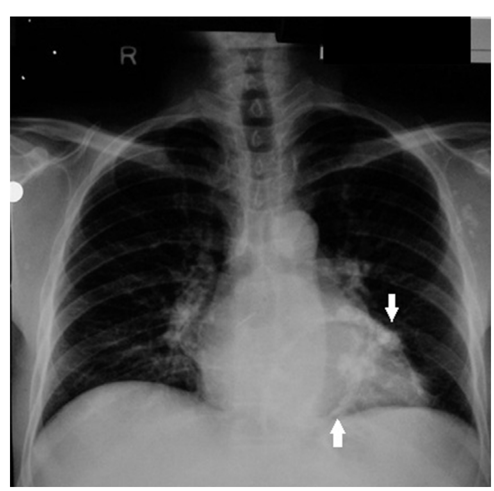

Fig. 4 – (A) Chest radiograph of a patient showing pericardial calcification primarily in the atrio-ventricular groove.

Table 3 – Comparison of echocardiography data before and after pericardiectomy in patients who responded to pericardiectomy.

Echocardiography parameters	Pre-pericardiectomy ($n = 20$)	Post-pericardiectomy ($n = 20$)	p value
Congested inferior vena cava	20	3	<0.001
Left atrium (mm)	39.33 ± 10.52	34.45 ± 10.08	<0.001
Left atrium:aortic annulus	1.93 ± 0.56	1.69 ± 0.54	<0.001
Septal bounce	20	4	<0.001
Ejection fraction (%)	60	60	
Tricuspid regurgitation (more than 1+)	1	1	
Mitral regurgitation (more than 1+)	1	1	
Pericardial effusion	9	0	<0.001
Pulmonary hypertension	0	0	
Mitral E velocity (cm/sec)			
Inspiratory	51.90 ± 15.63	70.30 ± 18.35	<0.001
Expiratory	77.2 ± 23.80	82.55 ± 21.56	0.46
Change (%)	39.23 ± 15.11	14.43 ± 7.76	<0.001
Tricuspid E velocity (cm/sec)			
Inspiratory	58.83 ± 8.64	58.15 ± 12.08	0.81
Expiratory	37.10 ± 7.90	47.75 ± 11.15	<0.001
Change (%)	31.33 ± 18.81	17.35 ± 16.26	<0.001
Mitral annular TDI (cm/sec)			
Medial e′	12.51 ± 1.94	10.72 ± 2.99	<0.001
Lateral e′	12.09 ± 3.01	12.78 ± 3.39	0.29
Medial e′:lateral e′ ratio	1.08 ± 0.27	0.87 ± 0.22	<0.03
Annular reversus	12	6	<0.001
Mitral E/medial e′	4.21 ± 1.35	6.91 ± 2.62	0.001

valve) without significant change in the maximal E velocities (i.e. during expiration at mitral valve and inspiration at tricuspid valve).

There was a significant respiratory variation of 39.23 ± 15.11% in the mitral E velocity before pericardiectomy, which was reduced to 14.43 ± 7.76% ($p < 0.001$) after surgery. Also there was a significant reduction in the respiratory variation in tricuspid E velocities from 31.33 ± 18.81% to 17.35 ± 16.26% ($p < 0.001$). One patient continued to demonstrate >25% respiratory variation in mitral inflow velocities.

The pre-operative septal (medial e′) and lateral (lateral e′) mitral annular velocities obtained by TDI were 12.51 ± 1.94 cm/sec and 12.09 ± 3.01 cm/sec, respectively. The mean ratio of medial e′ to lateral e′ was 1.03. 'Annulus reversus' (medial e′/lateral e′ > 1) was seen in 12 (60%) patients. Post-pericardiectomy, the medial e′ and lateral e′ were 10.72 ± 2.99 cm/sec ($p < 0.001$) and 12.78 ± 3.39 ($p > 0.29$), respectively. Thus, there was a significant fall in the medial e′, but the rise in lateral e′ was not significant. The mean ratio of medial e′ to lateral e′ reduced to 0.87 ($p < 0.03$). 'Annulus reversus' was found to persist in six (of 12 pre-operative) 'responders', i.e., the phenomenon resolved in 50% post-operatively ($p < 0.001$). The mitral E:medial e′ ratio (E/e′) was 4.21 ± 1.35 before pericardiectomy and increased to 6.91 ± 2.62 after surgery ($p = 0.0012$).

4. Discussion

CCP in developing countries is secondary to tuberculosis in 38–83% of cases.[4–7] In our study, 65% patients had biopsy-proven pericardial tuberculosis. None of our patients had collagen vascular disease or exposure to thoracic irradiation. Although constriction may follow the initial insult by as little as several months, it usually takes years to develop. CCP presents with predominant features of right heart failure. Signs and symptoms of elevated pulmonary venous pressures like exertional dyspnea, cough, and orthopnoea may develop with progressive disease. Echocardiography makes a significant contribution to the diagnosis. Impaired diastolic filling of the ventricles, enhanced ventricular interdependence and respiro-phasic dissociation of intracardiac pressures from intrathoracic pressures are evident.[8–10] Pericardiectomy is the standard treatment modality for symptomatic CCP. After pericardiectomy, 70–80% patients remain free from adverse cardiovascular outcomes at 5 years and 40–50% at 10 years. The operative mortality ranges from 5 to 15%.[11,12]

The mean patient age of 32.9 years seen in our study is lower than that seen in western studies. This may be accounted for by the higher prevalence of tubercular pericarditis in the younger population in India. The mean interval from onset of symptoms to pericardiectomy in this study was 12.3 ± 5.3 months, about 3–4 months higher compared to other studies. Even though tuberculous pericarditis is commonly seen, there is limited information regarding the role of corticosteroids. Though criticized for its analysis, the oft quoted South African trial in 240 patients of tubercular pericarditis reported a significant reduction in the need for repeat pericardiocentesis but not in the need for pericardiectomy at 24 months.[7,13,14] In our study, patients with pericardial effusion received oral corticosteroid therapy in addition to antitubercular drugs.

All patients underwent total pericardiectomy through midline approach. Total pericardiectomy is wide excision of the pericardium between the phrenic nerves, over the great vessels including the intrapericardial portion of and superior vena cava–right atrium (RA) junction and the diaphragmatic surface (including the IVC–RA junction). Any excision less than total is considered partial.[15] Total pericardiectomy is associated with lower perioperative and late mortality, and confers significant long-term advantage by providing superior hemodynamics that appear to be independent of the etiology of CCP.[15]

The CVP dropped from a mean of 20.7 ± 2.2 mm of Hg prior pericardiectomy to 12 ± 3.2 mm of Hg immediately post-procedure. CVP monitoring during pericardiectomy has been used as a surrogate marker for adequacy. However, the results of a study by Voila argue against the value of monitoring intracardiac pressures during decortication to assess the completeness of pericardial resection.[16] Clinical and hemodynamic responses to pericardiectomy may not always be dramatic and may continue over a variable period extending from a few weeks to months.[17–19] In all our patients, post-procedural echocardiography was done at least 3 months after the procedure.

The LA size reduced by a mean of 4.9 mm after successful pericardiectomy and thereby reflects significant improvement in diastolic properties of the heart. The ratio of LA size to aortic annulus size reduced significantly from 1.97 to 1.7 ($p < 0.001$) after surgery. The persistence of septal bounce, noted in a fourth of patients, was more common in those analyzed within 9 months of surgery and might resolve with time.

Since the heart needs to perform in a fixed and limited space in CCP, there is a significant reciprocal respiratory variation in the right and left ventricular filling measured in terms of mitral and tricuspid inflow velocities. Pericardiectomy improves filling dynamics as assessed by Doppler interrogation, although these may not parallel clinical improvement.[20] There was a rise in the minimal E velocities (i.e. during inspiration at mitral valve and expiration at tricuspid valve) without a significant change in the maximal E velocities (i.e. during expiration at mitral valve and inspiration at tricuspid valve). This may reflect a post-operative improvement in flow across the atrio-ventricular valves in those phases of the respiratory cycle, which had reduced flow pre-operatively (i.e. during inspiration at mitral valve and expiration at tricuspid valve). After pericardiectomy, there was loss of significant respiro-phasic (>25%) variation in the mitral E velocity in 19 (95%) patients. However, one patient with significant post-operative clinical improvement continued to demonstrate more than 25% respiratory variation in mitral E velocity. In a recently published study, pre-operative high early expiratory diastolic mitral inflow velocity more than 71 cm/sec predicted poor prognosis after surgery in CCP.[12] High mitral E velocity may reflect high LA pressures or associated myocardial involvement, thereby suggesting advanced disease. In our series, 11 (55%) of the 'responders' were noted to have expiratory mitral E velocity less than 71 cm/sec.

Use of TDI to assess the mitral annular velocity is a clinically useful tool in differentiating CCP from restrictive physiology. In CCP, early medial mitral annular diastolic velocity (septal e′) is preserved or increased as compared to early lateral mitral annular velocity (lateral e′). This is due to limitation of lateral annular expansion due to pericardial constriction. This phenomenon, described as 'annulus reversus', is relatively specific for CCP.[21] Annulus reversus was seen in only 60% patients prior to surgery in our study as compared to 74% in the study by Veress et al.[20] Interestingly, the phenomenon was found to resolve only in 50% of patients even after favorable clinical response to pericardiectomy. Pericardiectomy removes the constraint to lateral annular expansion. This nullifies the exaggerated longitudinal motion of the medial mitral annulus (medial e′). This may explain the subsequent significant reduction in medial e′ and a relatively small increase in lateral e′. In some cases, low annular velocities unmasked after pericardiectomy may reflect underlying myocardial involvement. This could be secondary to myocardial damage, atrophy secondary to longstanding encasement, penetration of the myocardium by the calcium spurs, inflammatory infiltration into myocardium, a firmly adherent pericardium or additional injury at the time of surgery.[21]

The E/e′ ratio is considered to be a surrogate marker for pulmonary capillary wedge pressure (PCWP).[22] The paradoxically inverse relationship between E/e′ and PCWP in CCP is termed as 'annulus paradoxus'.[22] The plausible explanation for this phenomenon is that in the presence of pericardial constriction, limited lateral expansion with subsequent exaggerated longitudinal motion of the medial mitral annulus results in a preserved or increased medial e′. Therefore, despite the higher filling pressures, the E/e′ ratio is low. In this study, invasive measurement of the PCWP was not performed. Notwithstanding, the significant post-operative rise in E/e′ from 4.21 to 6.91, presumably due to normalization of the e′, appears to indirectly confirm the phenomenon of annulus paradoxus.

4.1. *Observations in 'non-responders'*

The number of patients who did not respond to pericardiectomy ($n = 3$) in our study was inadequate to define clear predictors of outcome. However, certain features observed in all the three 'non-responders' were: pre-operative atrial fibrillation, larger mean LA size (38.9 mm), higher expiratory mitral E velocity (mean of 91.34) cm/sec), use of intraoperative CPB (2 of 3), persistent post-operative atrial fibrillation, persistent post-operative septal bounce and significant respiratory variation in mitral inflow velocities. The reasons described for persistent symptoms after surgery include imperfect or incomplete decortication and/or concomitant myocardial involvement.[23,24]

4.2. *Limitations*

The small sample size and lack of long-term follow-up must be acknowledged as major limitations of our study. Nevertheless, these observations present the largest data set published till date from western India. The comparison of echocardiographic data in patients may be confounded by age and the etiology of CCP. Although TDI was obtained from longitudinal axis motion in the 4-chamber view, analysis of multiple annular regions could have provided additional helpful data to understand radial and circumferential functions of the annulus. Also, respirometer gating and strain imaging would

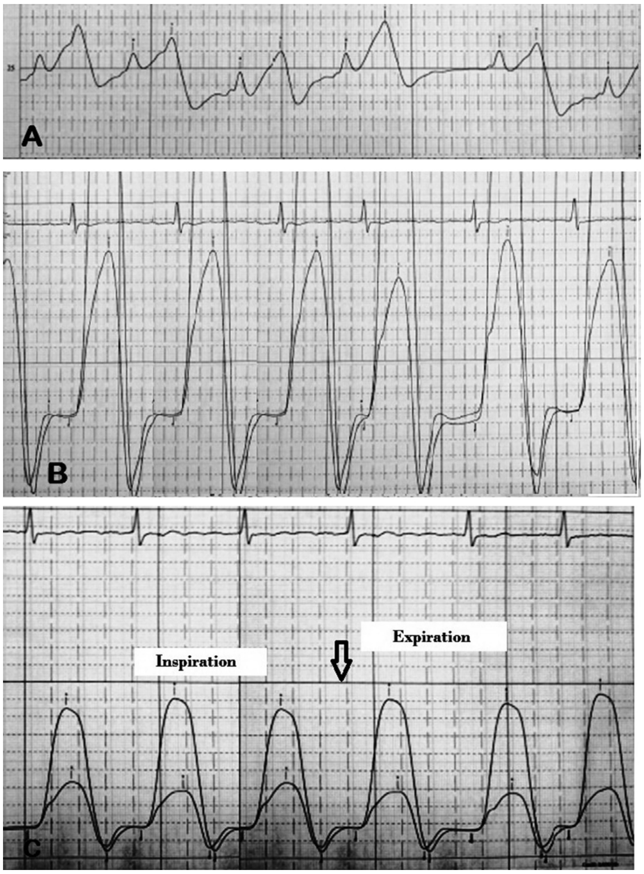

Fig. 5 – (A) Right atrial pressure tracings obtained after cardiac catheterization in a patient showing elevated pressures and prominent Y descent. Simultaneous pressure tracings obtained from both the ventricles showing (B) the typical 'square root' sign, equalization of diastolic pressures and (C) the 180° out of phase respirophasic variation in the systolic area under curve of both the ventricles, i.e. reduction in the right ventricular: left ventricular systolic area index in expiration.

have been desirable, but was not available. Cardiac catheterization (Fig. 5) was not done in all patients and therefore, the PCWP and the phenomenon of 'annulus paradoxus' could not be evaluated.

5. Conclusion

Echocardiography offers valuable insights into the physiology of pericardial constriction and the salutary effects of surgical pericardiectomy. Among the patients who showed clinical response to pericardiectomy, such evaluation revealed a significant reduction in vena caval congestion, LA size, ratio of LA: aortic annulus, septal bounce, respiratory variation in mitral and tricuspid E velocities, mitral annular medial e′ and the phenomenon of annulus reversus. Also, there was a significant rise in minimum tricuspid and mitral E velocities and the E/e′ ratio. These parameters should provide a useful framework for the assessment of patients of CCP after pericardiectomy.

Conflicts of interest

The authors have none to declare.

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